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**REPLY**

Auer et al. (1) investigated the relationship between subclinical hyperthyroidism and atrial fibrillation (AF), and they concluded that subclinical hyperthyroidism has a risk of AF. They also speculated that the thyroid hormone may have little effect on the genesis of AF in subclinical hyperthyroidism. We appreciated the comment from Auer and colleagues. However, in the study from Auer et al. (1), 65% of patients with AF were associated with other heart diseases (coronary artery disease, dilated cardiomyopathy and valvular heart disease), and about half of these patients had atrial enlargement. Thus, AF in these patients may be due to the combined effects of thyroid hormone and underlying heart diseases.

Subclinical hyperthyroidism refers to a state with normal thyroid hormone concentrations and low serum thyrotropin concentration. Several causes of subnormal thyrotropin do not reflect the presence of subclinical hyperthyroidism. Serum thyrotropin concentrations are frequently low in patients with severe non-thyroid illness, especially those receiving glucocorticoid. In addition, low serum thyrotropin value may be associated with low or normal serum thyroid hormone in the early stage or shortly after treatment or spontaneous resolution of overt hyperthyroidism (2). In the study from Auer et al. (1), all the patients had underlying thyroid diseases of functional autonomy or autoimmune. It is difficult to diagnose these patients from limited blood samplings, and subclinical hyperthyroidism may be overestimated.

Subclinical hyperthyroidism has been considered to have increasing risk of AF. In the follow-up of 10 years, there was a

threefold relative risk of AF in these patients (3). The negative feedback relationship between serum thyrotropin and thyroid hormone is log linear. Thus, the patients with slight excess of thyroid hormone would have low thyrotropin level, but would have serum thyroxine and tri-iodothyronine concentrations above the mean values for normal subjects but within the normal range. Patients with subclinical hyperthyroidism still have symptoms owing to the biologic effects of thyroid hormone. This means that patients with subclinical hyperthyroidism may still have excess thyroid hormone, and thyroid hormone can induce AF.

Although this experimental hyperthyroidism is not completely similar to usual hyperthyroidism, we investigated the direct effects of thyroid hormone on the electrophysiologic characteristics of pulmonary vein and atrial cardiomyocytes (4). We believe that thyroid hormone increases the arrhythmogenic activity of pulmonary vein cardiomyocytes, which may underlie the occurrence of AF in hyperthyroidism.

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## Manifestation of Left Main Coronary Artery Stenosis Is Diffuse ST Depression in Inferior and Precordial Leads on ECG

In the November 1, 2001, issue of the *Journal of the American College of Cardiology*, Yamaji et al. (1) reported on a novel electrocardiographic (ECG) sign for prediction of acute ischemia caused by left main coronary artery obstruction. They found that ST elevation in lead aVR with less ST elevation in lead V<sub>1</sub> is a predictor of left main obstruction. Searching the English-language literature they found only 42 previous reported patients with acute left main stenosis. The ECG description in these studies included right bundle branch block, anterior ST elevation or precordial ST